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Dr. Harker Replies

TO THE EDITOR: Thank you for the courtesy of giving us the opportunity to reply to the letter from Dr. Kudrow regarding increased platelet aggregability in association with migraine.

As pointed out in the introduction to the review by Dr. Huebsch and myself, we limited the discussion to clinically significant defects in platelet function, their pathophysiology, diagnosis and management. We did not include any disorders of increased platelet aggregability or the role of platelets in thrombosis or atherogenesis. Therefore, it would have been inappropriate to include a discussion on migraine.

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Accidental Hypothermia: Hypoglycemia or Hyperglycemia

TO THE EDITOR: We read with interest the article "Hypoglycemia and Accidental Hypothermia in an Alcoholic Population," in the August 1980 issue, which showed that 41 percent of Dr. Fitzgerald's hypothermic patients were hypoglycemic.[1] These data were surprising to those of us who have found the opposite results in our series of hypothermic patients. We are not sure that the incidence of hypoglycemia may be this high; in fact, in a review of 39 hypothermic patients, 34 of whom had impaired liver function or history of chronic alcoholism (or both) only 1 patient had a blood glucose level below 58 mg per dl.[2] All of our patients were hyperglycemic with glucose levels as high as 1,278 mg per dl.[3] None of our patients had a history or physical findings to suggest hepatic dysfunction. Hypothermia has multiple effects on the body's physiology. A recent review discusses the role of hypothermia producing hyperglycemia.[4]

We would suggest that there may be two dissimilar groups of hypothermic patients. One group may be characterized by the alcoholic patients with severe liver impairment and depleted glycogen stores with resultant impaired gluconeogenesis who therefore present with hypoglycemia. The other, and far larger group, would contain all those patients with relatively healthy livers who during their episodes of hypothermia may in fact be hyperglycemic to a pronounced degree. In her conclusions Dr. Fitzgerald suggested empirically treating hypothermic patients with an intravenous bolus of 50 percent glucose solution. We believe the use of a capillary blood glucose determination by a finger stick method in the emergency room would quickly and inexpensively identify these two groups. This would be preferable to empirically treating all hypothermic patients as having hypoglycemia.

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Dr. Fitzgerald Replies

TO THE EDITOR: Doctors Altus and Hickman express surprise at the high incidence of hypoglycemia in my alcoholic hypothermic patients. It was the same astonishment that led me to publish the series. Hypothermia may indeed lead to hyperglycemia in some patients. As tissue temperature falls, glucose consumption by cells decreases.[1,2] Pancreatic insulin release[3] and tissue sensitivity to insulin[4] may be blunted.

Four of our patients had an admission diagnosis of cirrhosis. One would expect, if the hypothesis forwarded by Doctors Altus and Hickman were correct, that these patients would be the most likely to be hypoglycemic. Yet in only one of these four was there a serum glucose value in the hypoglycemic range (37 mg per dl). The case has been made, in fact, that liver dysfunction may significantly contribute to hyperglycemia in hypothermic patients.[1] It is difficult to predict either from the published literature or the patient's past history whether or not a given hypothermic individual patient will be hyperglycemic or hypoglycemic. At the same time hypothermia may ob-